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Effects of Standing on the Induction of Paroxysmal Supraventricular Tachycardia

TAKAAKI HASHIMOTO, MD, MASAHIKO FUKATANI, MD, MITSUHIRO MORI, MD, KUNITAKE HASHIBA, MD, FACC

Nagasaki, Japan

To evaluate the effects of standing on induction of paroxysmal supraventricular tachycardia, electrophysiologic studies were performed in both the supine and standing positions in 22 patients with atrioventricular (AV) reciprocating tachycardia and in 11 with AV node reentrant tachycardia.

AV reciprocating tachycardia was induced in 9 of the 22 patients with AV reciprocating tachycardia when they were in the supine position and in 17 when standing. The effective refractory period of the AV node markedly shortened, from 275 ± 72 to 203 ± 30 ms ($n = 16$, $p < 0.005$) after standing. The effective refractory period of the accessory pathway shortened slightly, from 293 ± 75 to 278 ± 77 ms ($n = 8$, $p < 0.005$), after standing.

AV node reentrant tachycardia was induced in 3 of the 11 patients with AV node reentrant tachycardia when they were in the supine position and in 6 when standing. The effective refractory periods of the slow pathway and fast pathway shortened markedly, from 293 ± 72 to 216 ± 40 ms ($n = 6$, $p < 0.025$) and from 416 ± 85 to 277 ± 50 ms ($n = 10$, $p < 0.005$), respectively, after standing.

Plasma norepinephrine levels increased during standing both in patients with AV reciprocating and in those with AV node reentrant tachycardia ($n = 11$, $p < 0.005$, $n = 8$, $p < 0.005$, respectively).

In conclusion, standing, which is associated with increased sympathetic tone, changed the electrophysiologic properties of the reentrant circuits, facilitating induction of AV reciprocating tachycardia and AV node reentrant tachycardia.

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Paroxysmal supraventricular tachycardia is well known to occur during exercise and to terminate on rest in the supine position (1–4). The mechanisms of exercise-induced supraventricular tachycardia are difficult to identify. These may involve an increase in the number of spontaneous premature beats during exercise or changes in the electrophysiologic properties of the reentry pathways, or a combination of both, which facilitate initiation of supraventricular tachycardia during the normal sinus tachycardia produced by exercise.

To clarify the effects of standing on induction of paroxysmal supraventricular tachycardia, electrophysiologic studies were performed before and after standing in 33 patients with spontaneous episodes of paroxysmal supraventricular tachycardia.

Methods

Study patients. The subjects in this study were 33 consecutive patients with spontaneous episodes of paroxysmal supraventricular tachycardia. All had undergone electrophysiologic studies and had a diagnosis of atrioventricular (AV) reciprocating tachycardia or AV node reentrant tachycardia at Nagasaki University Hospital between October 1987 and September 1989.

A standard electrophysiologic study had been performed in the cardiac catheterization laboratory 1 day before this study. AV reciprocating tachycardia using the AV node for anterograde conduction and the accessory pathway for retrograde conduction was diagnosed in 22 patients; this tachycardia was persistent as a result of Wolff-Parkinson-White syndrome in 8 patients, intermittent in 5 and concealed in 9 of 12 male and 10 female patients, mean age 41 years, range 17 to 62. AV node reentrant tachycardia using the slow pathway for anterograde conduction and the fast pathway for retrograde conduction was diagnosed in 11 patients (5 male and 6 female, mean age 53 years, range 16 to 63).

All patients gave informed consent to the study, which was approved by the Regional Ethics Committee in 1987.

Electrophysiologic studies. After the standard electrophysiologic study in the cardiac catheterization laboratory, an atrial J-shaped temporary pacing lead with quadripolar electrodes was introduced percutaneously into the right subclavian vein and placed, under fluoroscopy, in the appendage of the right atrium. The distal pair of electrodes was used for electrical stimulation and the proximal pair for recording.

Electrophysiologic studies were performed with the patient supine and standing in the laboratory located in the

From the Third Department of Internal Medicine, Nagasaki University School of Medicine, Nagasaki, Japan.

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Address for reprints: Takaaki Hashimoto, MD, The Third Department of Internal Medicine, Nagasaki University School of Medicine, 7-1 Sakamoto-cho, Nagasaki 852, Japan.

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Table 1. Summary of Data Before and After Postural Change for the 22 Patients With AV Reciprocating Tachycardia

	Supine	Standing	Supine
CL of tachycardia (ms)	363 ± 52 (n = 11)	307 ± 37 (n = 11)	343 ± 35 (n = 6)
AV interval during tachycardia (ms)	225 ± 58 (n = 11)	175 ± 45 (n = 11)	205 ± 23 (n = 6)
VA interval during tachycardia (ms)	139 ± 21 (n = 11)	132 ± 20 (n = 11)	138 ± 23 (n = 6)
Heart rate (beats/min)	75 ± 11 (n = 22)	94 ± 16 (n = 22)	80 ± 14 (n = 12)
Systolic BP (mm Hg)	123 ± 14 (n = 22)	111 ± 10 (n = 22)	122 ± 17 (n = 12)
Diastolic BP (mm Hg)	75 ± 9 (n = 22)	81 ± 8 (n = 22)	77 ± 12 (n = 12)

AV = atrioventricular; BP = blood pressure; CL = cycle length; VA = ventriculoatrial.

patient's ward on the afternoon of the day after the standard electrophysiologic study that was performed in the cardiac catheterization laboratory. Patients assumed the supine position and a blood pressure cuff was applied to the left arm. A 20 gauge needle catheter was introduced into an antecubital vein of the right arm and attached to a three-way stopcock. The stopcock-catheter assembly was filled with a sterile saline solution, which dripped continuously to maintain catheter patency. The right atrial electrogram as well as electrocardiographic (ECG) leads I, aVF, and V_1 were simultaneously displayed on a multichannel oscilloscope and recorded directly on an ink-jet recorder at a paper speed of 100 mm/s.

After the needle catheter was placed, patients were permitted to rest quietly in the supine position for 20 min. At the end of that time, heart rate and blood pressure were measured and 5 ml of blood was withdrawn through the three-way stopcock.

Electrophysiologic study was performed using a programmed digital stimulator which delivered impulses of 1.5 ms at approximately twice diastolic threshold. Atrial premature stimulation was used to determine the effective refractory periods of the AV node and accessory pathway at a basic cycle length of 500 ms in 28 patients and 700 ms in the 5. A premature atrial beat was introduced after every eighth paced beat until atrial refractoriness was reached, and the premature stimulus was scanned in steps of 10 ms. Atrial incremental pacing was performed to induce supraventricular tachycardia if it was not induced with atrial premature stimulation. Atrial incremental pacing was performed up to the onset of 2:1 AV block. Patients were then asked to rise and stand in place for 10 min. Heart rate and blood pressure were again recorded, a second blood sample was withdrawn, and the electrophysiologic study was repeated. Twenty consecutive patients of the entire series of 33 patients were then asked to lie supine and, after 10 min, a third blood sample was withdrawn and the electrophysiologic study was repeated. All procedures were completed without any complications.

Plasma norepinephrine levels were measured by high performance liquid chromatography with fluorescence detection. All antiarrhythmic medications had been discontinued for at least 5 half-lives before the study.

Definition of terms.

Supraventricular tachycardia. Supraventricular tachycardia that lasted >30 s.

Short run of supraventricular tachycardia. At least two consecutive atrial echo beats, but <30 s of supraventricular tachycardia.

Atrial echo beat. An atrial echo beat induced by the same mechanism as the supraventricular tachycardia.

Anterograde effective refractory period of the AV node. The longest atrial coupling interval (A_1A_2 interval) in which A_2 was blocked in the AV node, where A_1 represents an atrial electrogram of the basic atrial drive beats and A_2 an atrial electrogram of the atrial premature stimulation.

Anterograde effective refractory period of the accessory pathway. The longest atrial coupling interval in which A_2 was blocked in the accessory pathway.

Anterograde effective refractory period of the slow pathway. The longest atrial coupling interval in which A_2 was blocked in the slow pathway of the AV node.

Anterograde effective refractory period of the fast pathway. The longest atrial coupling interval in which A_2 was blocked in the fast pathway of the AV node.

Functional refractory period of the atrium. The shortest A_1A_2 interval in response to any S_1S_2 interval, where S_1 represents a stimulus artifact of the basic atrial drive beats and S_2 a stimulus artifact of the premature stimulus.

Jump from the fast pathway to the slow pathway. At least 40 ms of prolongation of the AV interval, with a 10 ms decrease in the coupling interval of the atrial premature stimulus.

Statistical analysis. Results are presented as mean values ± SD. The paired *t* test was used to compare group means. A $p < 0.05$ was considered to indicate a significant difference.

Results

At the time of this study all patients had normal sinus rhythm. The values of electrophysiologic measurements were used only when they were obtained in both the supine and standing positions in a patient.

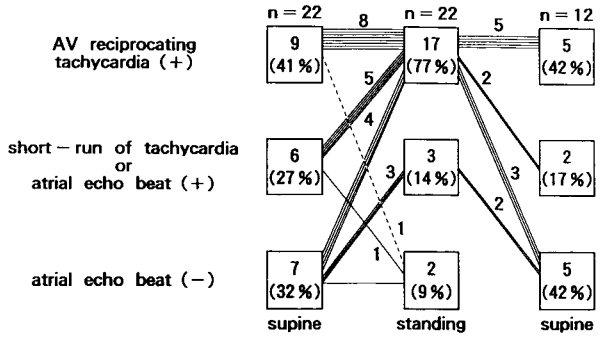

Figure 1. Results of induction of atrioventricular (AV) reciprocating tachycardia, short runs of tachycardia or atrial echo beats induced by atrial premature stimulation before and after postural change in the 22 patients with AV reciprocating tachycardia. **Dotted line** indicates a patient with dual AV pathways.

AV Reciprocating Tachycardia (Table 1)

Induction of AV reciprocating tachycardia (Fig. 1). In the supine position, AV reciprocating tachycardia was induced in 9 of the 22 patients (41%) and short runs of AV reciprocating tachycardia or atrial echo beats were induced in six (27%). AV reciprocating tachycardia was induced after standing in 17 patients (77%) and short runs of AV reciprocating tachycardia or atrial echo beats in 3 (14%). After the supine position was reassumed, AV reciprocating tachycardia was induced in 5 (42%) of 12 patients and short runs of AV reciprocating tachycardia or atrial echo beats were induced in 2 (17%).

In one patient with Wolff-Parkinson-White syndrome and dual AV node pathways, AV reciprocating tachycardia was induced only when an impulse was blocked in the refractory fast pathway and proceeded along the slow pathway in the supine position. After standing, a jump from the fast pathway to the slow pathway failed to occur, because the effective refractory period of the fast pathway had been markedly shortened to less than that of the slow pathway. Therefore, AV reciprocating tachycardia could not be induced after standing.

Anterograde effective refractory periods of the AV node and accessory pathway (Fig. 2). The effective refractory periods of the AV node were determined in 16 of the 22 patients in both the supine and the standing position. If the functional refractory period of the atrium was reached before AV block occurred, this period was used to estimate the effective refractory period of the AV node.

The mean effective refractory period of the AV node shortened from 275 ± 72 to 203 ± 30 ms ($n = 16$, $p < 0.005$) after standing, and was 262 ± 90 ms ($n = 9$, $p < 0.005$) after the supine position was reassumed (Fig. 2).

The effective refractory period of the accessory pathway, measured for all eight patients with manifest Wolff-Parkinson-White syndrome in both the supine and standing positions, shortened from 293 ± 75 to 278 ± 77 ms ($n = 8$, $p < 0.005$) after standing, and was 273 ± 15 ms ($n = 3$, $p < 0.05$) after the supine position was reassumed (Fig. 2).

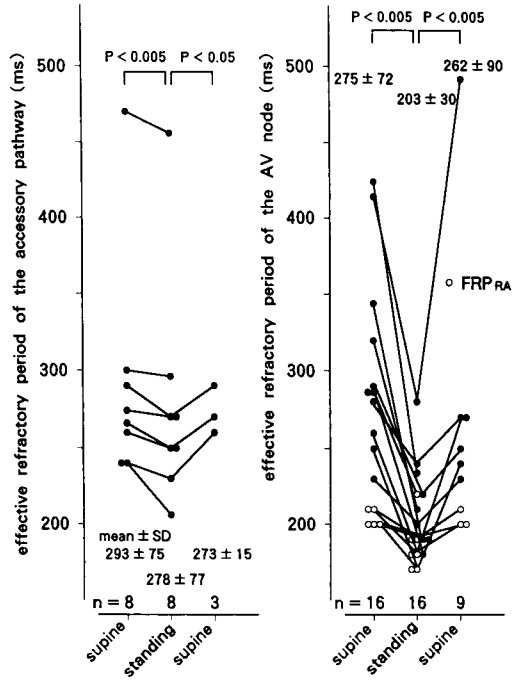

Figure 2. Changes in the anterograde effective refractory period of the accessory pathway (**left**) and AV node (**right**) after postural change for the 22 patients with AV reciprocating tachycardia. **Open circles** indicate functional refractory period of the atrium (FRPRA) and **solid circles** the anterograde effective refractory period of AV conduction.

In the four patients with manifest Wolff-Parkinson-White syndrome in whom AV reciprocating tachycardia was induced only while standing, the effective refractory period of the AV node, which had been longer than that of the accessory pathway, became shorter after standing. This shortening permitted retrograde conduction up the accessory pathway to the atrium, causing an echo beat and tachycardia. In the other five patients with intermittent or concealed Wolff-Parkinson-White syndrome, in whom AV reciprocating tachycardia was induced only during standing, the effective refractory period of the AV node shortened sufficiently to initiate and perpetuate supraventricular tachycardia.

Cycle length of AV reciprocating tachycardia. In the 11 patients in whom AV reciprocating tachycardia was induced by atrial premature or incremental pacing in both the supine and the standing position, the cycle length significantly shortened after standing ($p < 0.005$). This shortening was mainly the result of shortening of the AV interval during the tachycardia (Table 1).

Heart rate, blood pressure and plasma norepinephrine. Diastolic blood pressure ($p < 0.01$) and heart rate ($p < 0.005$) increased and systolic blood pressure decreased ($p < 0.005$) after standing ($n = 22$) (Table 1). Plasma norepinephrine was measured in 11 consecutive patients with AV reciprocating

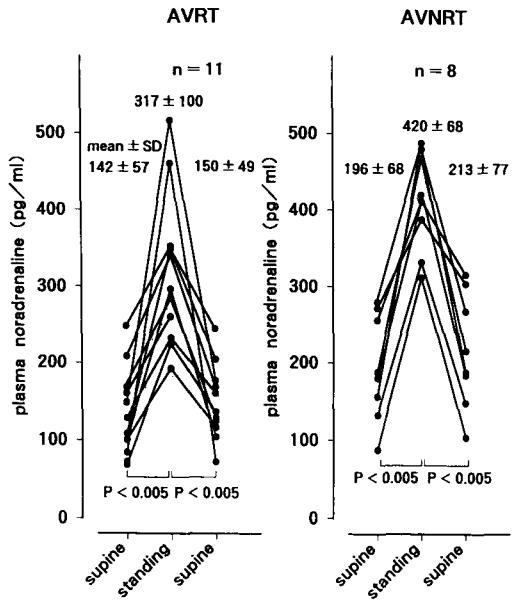

Figure 3. Changes in plasma norepinephrine (noradrenaline) levels after postural change for the 11 patients with atrioventricular reciprocating tachycardia (AVRT) (**left**) and for the patients with atrioventricular node reentrant tachycardia (AVNRT) (**right**).

tachycardia; it was significantly increased, from 142 ± 57 to 317 ± 100 pg/ml ($p < 0.005$), after standing, and returned to 150 ± 49 pg/ml ($p < 0.005$) after the supine position was reassumed (Fig. 3).

AV Node Reentrant Tachycardia (Table 2)

Induction of AV node reentrant tachycardia (Fig. 4). AV node reentrant tachycardia was induced in 3 (27%) of the 11 patients in the supine position, and short runs of AV node reentrant tachycardia or atrial echo beats were induced in three (27%) (Fig. 4). On standing, AV node reentrant tachycardia was induced in six patients (55%) and short runs or atrial echo beats were induced in one patient (9%). After the supine position was reassumed, AV node reentrant tachycardia was induced in one (13%) of eight patients and short runs or atrial echo beats in three (38%).

Anterograde effective refractory periods of the slow pathway and the fast pathway (Fig. 5). The effective refractory period of the slow pathway was determined in 6 patients and that of the fast pathway in 10 of the 11 patients in both the

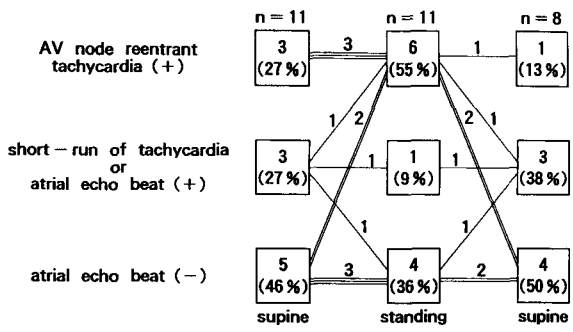

Figure 4. Results of the induction of atrioventricular (AV) node reentrant tachycardias, short runs of tachycardia or atrial echo beats induced by atrial premature stimulation before and after postural change in the 11 patients with AV node reentrant tachycardia.

supine and the standing position. If the functional refractory period of the atrium was reached before AV block occurred, this period was used to estimate the effective refractory period of the AV node.

The mean effective refractory periods of the slow pathway and the fast pathway shortened from 293 ± 72 to 216 ± 40 ms ($n = 6$, $p < 0.025$) and from 416 ± 85 to 277 ± 50 ms ($n = 10$, $p < 0.005$), respectively (Fig. 5). Values after reassuming the supine position were, respectively, 295 ± 44 ($n = 4$, $p < 0.01$) and 421 ± 83 ms ($n = 7$, $p < 0.005$).

In two patients, a jump from the fast pathway to the slow pathway failed to occur in the supine position, but AV node reentrant tachycardia was initiated and perpetuated while the patient was standing. This alteration occurred because the effective refractory period of the slow pathway, which had been longer than that of the fast pathway, shortened to less than that of the fast pathway after standing. In the other two patients in whom AV node reentry was induced only before standing, the effective refractory period of the fast pathway, which had been longer than that of the slow pathway, shortened to less than that of the slow pathway after standing.

Cycle length of AV node reentrant tachycardia. AV node reentrant tachycardia was induced by atrial premature or incremental pacing in 3 of the 11 patients in both the supine and the standing position. The cycle length of the tachycardia significantly shortened after standing ($p < 0.01$), and this shortening was mainly the result of shortening of the AV interval during the tachycardia (Table 2).

Table 2. Summary of Data Before and After Postural Change for the 11 Patients With Atrioventricular Node Reentrant Tachycardia

	Supine	Standing	Supine
CL of tachycardia (ms)	378 ± 81 (n = 3)	323 ± 83 (n = 3)	375 (n = 1)
AV interval during (ms) tachycardia	335 ± 74 (n = 3)	278 ± 73 (n = 3)	335 (n = 1)
VA interval during (ms) tachycardia	43 ± 14 (n = 3)	42 ± 12 (n = 3)	40 (n = 1)
Heart rate (beats/min)	77 ± 11 (n = 11)	94 ± 16 (n = 11)	76 ± 12 (n = 8)
Systolic BP (mm Hg)	134 ± 17 (n = 11)	121 ± 22 (n = 11)	130 ± 17 (n = 8)
Diastolic BP (mm Hg)	79 ± 18 (n = 11)	88 ± 16 (n = 11)	90 ± 16 (n = 8)

Abbreviations as in Table 1.

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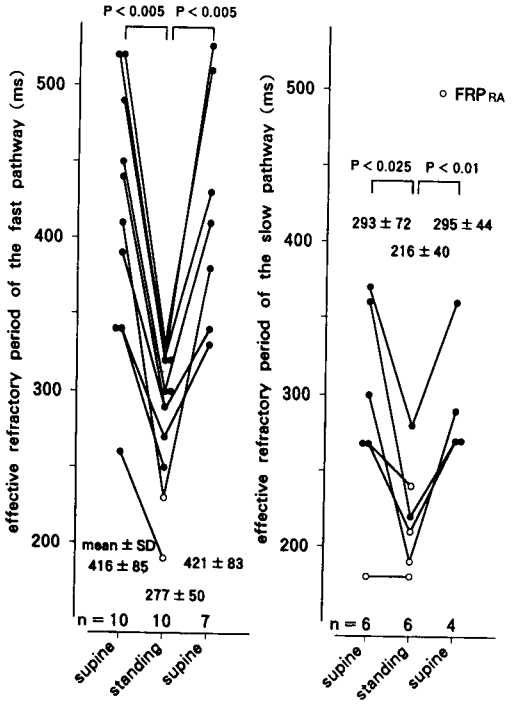

Figure 5. Changes in the anterograde effective refractory period of the fast pathway (**left**) and slow pathway (**right**) after postural change for the 11 patients with atrioventricular (AV) node reentrant tachycardia. **Open circles** indicate functional refractory period of the atrium (FRPRA) and **solid circles** anterograde effective refractory period of AV conduction.

Heart rate, blood pressure and plasma norepinephrine. Diastolic blood pressure ($p < 0.01$) and heart rate ($p < 0.005$) increased and systolic blood pressure decreased ($p < 0.025$) after standing ($n = 11$) (Table 2). Plasma norepinephrine, measured in eight consecutive patients with AV node reentrant tachycardia, increased from 196 ± 68 to 420 ± 68 pg/ml ($p < 0.005$) after standing; it returned to 213 ± 77 pg/ml ($p < 0.005$) after the supine position was reassumed (Fig. 3).

Discussion

The importance of the sympathetic nervous system in the genesis of paroxysmal supraventricular tachycardia has been suggested by clinical observation and exercise tests (1,2,5). The role of adrenergic effects, which modify the refractory period of the reentrant circuit, has been mainly investigated by electrophysiologic studies before and after administration of isoproterenol (6–12). Brugada et al. (6) reported that the effects of isoproterenol are independent of the relation between spontaneous episodes of AV reciprocating tachycardia and exercise. The same results were reported for ventricular tachycardia (13). The effects of isoproterenol and exercise on the facilitation of paroxysmal supraventricular tachycardia do not always coincide (6,7) because changes in autonomic tone induced by isoproterenol mimic but are not equivalent to the changes in autonomic tone induced by exercise.

Postural change and intrinsic sympathetic tone. Standing is a simple and useful way to activate sympathetic tone (14,15). Increases in the plasma concentration of norepinephrine are believed to result from, and to reflect an activation of, the sympathetic nervous system (14–22).

Electrophysiologic mechanisms of facilitation of AV reciprocating tachycardia by standing. Brugada et al. (6) observed that isoproterenol facilitated induction of AV reciprocating tachycardia, mainly by facilitating anterograde conduction across the AV node. In our previous study (8), isoproterenol also facilitated induction of AV reciprocating tachycardia, mainly by shortening the effective refractory period of the AV node. A similar finding was reported in five patients with exercise-related AV reciprocating tachycardia after isoproterenol administration (7).

Standing, which activates sympathetic tone, altered the electrophysiologic properties sufficiently to initiate and perpetuate tachycardia: 1) the anterograde effective refractory period of the AV node was shorter than that of the accessory pathway; and 2) the anterograde effective refractory period of the AV node was shortened enough to permit continuous reciprocation (23).

Denes et al. (24) showed that the occurrence of AV reciprocating tachycardia depends not only on adequate anterograde AV node conduction, but also on the capability for retrograde anomalous pathway conduction. Retrograde conduction was not measured in our study.

Electrophysiologic mechanisms of facilitation of AV node reentrant tachycardia by standing. The relative change in the effective refractory periods of the slow and fast pathways after postural change was more complex than the relative change in the effective refractory periods of the AV node and the accessory pathway. Standing, associated with an increased plasma catecholamine level, may facilitate but may also prevent the initiation of AV node reentrant tachycardia. Findings were similar (9,25,26) in patients in whom AV node reentrant tachycardia could not be induced after isoproterenol or atropine administration.

Huycke et al. (9) showed that isoproterenol-facilitated induction of AV node reentrant tachycardia was mediated by improved conduction in the anterograde slow pathway in two of six patients, in the retrograde fast pathway in three, and in both the anterograde slow and retrograde fast pathways in one patient. Hariman et al. (10) reported that catecholamines allowed induction of AV node reentrant tachycardia by enhancement of retrograde fast conduction, but these observations were not confirmed in our study.

Effect of standing on cycle length of supraventricular tachycardia. Brugada et al. (6) and Wellens et al. (11) showed that the shortening of the supraventricular tachycardia cycle length by isoproterenol was mainly the result of a shortening in the AH interval during the tachycardia. Our result is consistent with their data.

Clinical implications. Standing can be used to induce paroxysmal supraventricular tachycardia and to clarify the

effects of intrinsic sympathetic activity on its initiation and perpetuation.

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